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Good Nutritional Control May Prevent Polyneuropathy after Bariatric Surgery

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Abstract

Introduction—Previously we showed that peripheral neuropathy occurs after bariatric surgery and was associated with malnutrition (mainly sensory polyneuropathy). This study asks whether a multidisciplinary approach to bariatric surgery lowers risk for developing peripheral neuropathy.

Methods—We performed a retrospective cohort study of all patients with bariatric surgery at Mayo Clinic between 1985 and 2002. Patients underwent intensive nutritional management before and after surgery. Potential risk factors were analyzed using life-table methods (Cox regression).

Results—Univariate analysis showed the following risk factors: increased serum glycosylated hemoglobin and triglycerides, prolonged hospitalization, postoperative gastrointestinal symptoms, nausea and vomiting. Peripheral neuropathy occurred less frequently (7% vs. 13%, $p < 0.01$) and specifically the sensory polyneuropathy subtype (1% vs. 7%, $p < 0.0001$) than in our prior cohort.

Discussion—A systematic, multidisciplinary approach of intensive nutritional management before and after surgery with frequent follow-up greatly decreased development of peripheral neuropathy (especially sensory polyneuropathy) in patients receiving bariatric surgery.

Keywords

bariatric surgery; peripheral neuropathy; nutritional deficiency; sensory polyneuropathy; hypertriglyceridemia

INTRODUCTION

Bariatric surgery (BS) is increasingly performed for morbid obesity. Criteria for BS include a body mass index (BMI) $> 40 \text{ kg/m}^2$ or a BMI $> 35\text{--}40 \text{ kg/m}^2$ with accompanying comorbidity (hypertension, diabetes mellitus [DM], etc.).¹ Neurologic complications of BS include peripheral neuropathy (PN), burning feet syndrome, meralgia paresthetica, posterolateral myelopathy, myotonic syndrome, Wernicke-Korsakoff encephalopathy, optic neuropathy, and lumbosacral plexopathy.^{2–7}

Previously, we conducted a large, case-controlled study of PN in a cohort of patients with morbid obesity who had BS performed at many different centers and were seen subsequently at Mayo Clinic.⁸ Our study showed that PN occurred more frequently after BS (16%) than after another open abdominal operation (3%) and that the three observed clinical patterns were mononeuropathy, sensory-predominant polyneuropathy, and radiculoplexus neuropathy. Risk factors for PN included rate and absolute amount of weight loss, prolonged gastrointestinal (GI) symptoms, failure to attend a nutritional clinic after BS, decreased serum albumin and transferrin levels after BS, postoperative surgical complications requiring hospitalization, jejunioileal bypass, and performance of the procedure at a non-Mayo center.⁸ We also found that the sensory-predominant subtype was highly associated with nutritional deficiencies, whereas the mononeuropathies and radiculoplexus neuropathies were not. However, the patients in our previous study had a variety of surgical settings and different degrees of follow-up. We decided that it was important to study a large cohort of patients who underwent BS at one tertiary referral center (Mayo Clinic), had a standardized, long term follow-up and attended a nutritional clinic pre- and postoperatively to see if the frequency and risk factors for development of PN after BS would be altered. We hypothesized that with well-controlled and regulated nutritional status, the occurrence of sensory-predominant polyneuropathy should be reduced.

MATERIALS AND METHODS

We used Mayo Clinic Health Science Research coded diagnoses to identify the medical records of all patients who underwent BS at Mayo Clinic in Rochester, MN from 8/1985 to 1/2002 and developed PN after BS. To be included in the study, a patient underwent an open BS for the indication of morbid obesity. All patients had BS, and pre- and postoperative evaluations were done at Mayo Clinic. A patient was excluded if 1) BS was done for an indication other than obesity, 2) PN was already present at the time of BS, 3) the follow-up period was less than one year, 4) the medical record was incomplete, or 5) the patient had a systemic disease that affects weight. Patients were not excluded because they had DM. Data from the medical record were collected from the time of preoperative evaluation for BS until the last follow-up visit. The definitions of PN, BS, weight measurements, neuropathic evaluations, laboratory methods, and nutritional management were the same as we used in our previous study.⁸ Nutritional management recommended included scheduled dietitian visits before surgery and 6 weeks, 3 months, 6 months, 12 months and annually after surgery. Patients received advice on dietary changes to promote weight loss and assure they were meeting nutritional needs. Vitamin and mineral supplementation included 2 multivitamin and mineral preparations daily, calcium supplement (> 1500 mg elemental calcium daily) and Vitamin B12, 1,000 mcg SQ monthly. Laboratory parameters were monitored to assess the need for additional supplementation (iron, Vitamin D).⁹ Only 75 patients were included in both studies. When we made direct comparisons between groups, we subtracted these 75 overlapping patients from the original cohort in order to not compare patients against themselves (this provided Mayo vs. non-Mayo BS cohorts).

Statistical analysis

Direct comparison of baseline characteristics was done using the Fisher exact test for dichotomous data and Student's t-test for continuous data. Data were then analyzed using univariate and multivariate life-table methods (Cox regression) to identify the risk factors associated with PN. In this method, the data were compared at baseline (time of BS) between patients who developed PN and all others who did not develop PN. At each follow-up time period, the data of patients who developed PN were compared to all other patients who did not have PN at that time (within 6 months before or after) and who were still being followed. The following variables were analyzed using life-table methods (at baseline and

over time, along with transformations of some variables): age, sex, DM at the time of BS, type of DM, height, weight, BMI, serum levels of albumin, B12, folate, glucose, glycosylated hemoglobin, hemoglobin, total cholesterol, triglycerides, and thyroid stimulating hormone (TSH), and blood iron, iron saturation, total iron binding capacity (TIBC), and total lymphocyte count. In addition, type of BS (Roux-en-Y gastric bypass, vertical banded gastroplasty and partial pancreatobiliary bypass), duration of hospital stay, time to onset of neuropathy, postoperative complications requiring hospitalization, prolonged post-op gastrointestinal (GI) symptoms (diarrhea, dumping, nausea/vomiting, other), postoperative nutrition (full, partial, none), and postoperative nutritional clinic attendance were also analyzed. A value of $p < 0.05$ was used for significance. Continuous data are reported as mean $\pm$ standard deviation.

RESULTS

Seven hundred and sixty-four patients had BS at Mayo Clinic between 8/1985 and 1/2002. Patients were excluded for the following reasons: 28 had BS performed for an indication other than morbid obesity; 35 had incomplete records; 64 had unavailable medical records; 61 developed PN before BS; 177 had follow-up performed elsewhere or the total follow-up time was less than one year; and 6 had severe systemic illnesses that could affect weight.

There were 393 patients who were included and followed up in this cohort. There were 284 women (72%) and 109 men (28%). One hundred and five (27%) patients had DM at the time of surgery. The follow-up time was a median of 26 months (range 12-223 months). The bariatric procedures included Roux-en-Y gastric bypass (320 patients), vertical banded gastroplasty (64), and pancreatobiliary bypass (9). Seven patients had a previous jejunoileal bypass that was converted to another type of BS. After BS, all patients were followed at Mayo Clinic; 351 (91%) patients were seen in the nutritional clinic, while the others were followed by their primary physician. Three hundred and forty-one (90%) received one or more forms of nutritional supplements after BS. Most of those who did not receive nutritional supplements had vertical banded gastroplasty, which usually does not require nutritional supplementation. Prolonged postoperative GI symptoms occurred in 30 (8%) patients including persistent (more than 1 month) nausea and vomiting (14 patients), dumping syndrome (2), diarrhea (13), and other (3). Eleven (3%) developed postoperative surgical complications that required re-hospitalization. The weight loss for the whole group after BS was 43 ± 25 kg (range -31 to 159).

Twenty-six (7%) patients developed PN after BS at a median time of 16 months after BS (range 1-108 months). The same three patterns of neuropathy occurred that we observed previously: mononeuropathy (21 patients, 81% of total PN identified), sensory-predominant polyneuropathy (2, 8%), and radiculoplexus neuropathy (3, 11%). Mononeuropathies included 16 patients with median mononeuropathy at the wrist (carpal tunnel syndrome), 2 patients with superficial radial sensory neuropathies, and one patient each with lateral femoral cutaneous neuropathy (meralgia paresthetica), ulnar neuropathy at the elbow, peroneal neuropathy, and greater occipital neuropathy. One patient had both carpal tunnel syndrome and superficial radial sensory neuropathy. Two patients had lumbosacral, and one had cervical radiculoplexus neuropathy.

Using t-tests and Fisher exact tests, we compared baseline characteristics of patients who developed PN to those who did not (Table 1). Increased blood glycosylated hemoglobin levels at baseline, longer duration of hospital stay, prolonged postoperative GI symptoms, and a lesser rate of attendance at nutritional clinics were significantly associated with developing PN after BS ($p < 0.04$ each).

Using life-table analyses, we compared data at baseline and at each follow-up time period in patients who developed PN to those who did not develop PN and identified several risk factors (Table 2). Risk factors for any PN after BS included: increased baseline glycosylated hemoglobin, increased serum triglycerides at follow-up, longer duration of hospital stay, prolonged postoperative GI symptoms, and prolonged postoperative nausea and vomiting. Risk factors for mononeuropathy after BS included: increased baseline glycosylated hemoglobin and longer duration of hospital stay. Risk factors for PN after BS in diabetic patients included: total lymphocyte count at follow-up, increased baseline glycosylated hemoglobin, increased serum triglycerides at follow-up and postoperative complications that required hospital admission. Risk factors for PN after BS in non-diabetic patients included: increased total lymphocyte count at follow-up and prolonged duration of hospital stay.

Because of the low prevalence of PN, we did not have the power to do multivariate analyses. In this study in contrast to our prior study, the amount of weight loss, change in BMI, serum albumin level, type of BS, and postoperative attendance at our nutritional clinic were not associated with PN.

We also compared the occurrence of PN and other baseline characteristics between the new cohort (Mayo BS patients) and the old cohort (non-Mayo BS patients). We found that the new cohort had lower rates of patients who had postoperative PN (7% vs. 13%, $p<0.01$). There were no significant differences in age or gender between the two groups. Mononeuropathies (5% vs. 5%, $p>0.05$) and radiculoplexus neuropathies (1% vs. 1%, $p>0.05$) occurred at similar rates, but the sensory-predominant polyneuropathies were markedly reduced in the new cohort (1% vs. 7%, $p<0.0001$) (Table 3).

DISCUSSION

We previously showed that peripheral neuropathy can occur after bariatric surgery and that risk factors related to poor nutritional status. This study is different and more rigorous in that we used univariate and multivariate regression analysis and systematically followed a cohort of people who had bariatric surgery performed at one institution. The hypothesis of this study was that an intensive, multidisciplinary, pre- and postoperative program of nutritional and surgical care at one institution would alter or prevent the development of PN after BS. To accomplish this, we looked at patients who had BS performed at our tertiary center where we have a multidisciplinary approach including nutritional supplementation. We found that the rate of PN after BS was decreased but not eliminated (26 of 393, 7%). These findings are in contrast to our previous cohort of non-Mayo BS patients (where BS was performed in various practice settings with less intense nutritional surveillance) in which PN after BS occurred in 45 of 360 cases (13%, $p<0.01$).⁸ The lesser rate of PN after BS can probably be explained by more intensive pre- and postoperative nutritional management and by fewer surgical complications. In contrast to the previous study, patients in this study: 1) had regular, multidisciplinary follow-up at our institution for at least one year, and almost all (91%) were followed in our nutritional clinic; 2) received some form of nutritional supplementation (90%); 3) had a lesser rate of postoperative GI symptoms (8% vs. 35% in the prior study); and 4) had fewer postoperative surgical complications that required hospitalization (3% vs. 27% in the prior study).

Almost all PN that developed in this cohort were mononeuropathies (21, 81%) with only small numbers of sensory-predominant polyneuropathy (2, 8%) and radiculoplexus neuropathy (3, 11%); these distributions are different from what we found previously.⁸ In the earlier study, we showed that PN after BS was associated with poor nutritional status, and the nutritional deficiencies were associated primarily with the sensory-predominant polyneuropathy subtype. Consequently, we hypothesized that if we eliminated the

nutritional deficiencies and avoided a too rapid weight loss in a cohort of BS patients that the occurrence of sensory-predominant polyneuropathy would decrease. We studied this question in this cohort and confirmed our hypothesis by finding a marked reduction in the number of cases of sensory-predominant polyneuropathy (1% vs. 7%) (Table 3). Therefore, because this cohort had much better postoperative follow-up in nutritional clinics, lost weight in a more controlled fashion, received more nutritional supplementation, had less prolonged GI symptoms, and had a much lower rate of postoperative complications, they did not become malnourished and did not develop a sensory predominant polyneuropathy. We acknowledge that, since the present cohort had their operations performed at one center and the previous cohort had their operations at many different centers, some of the differences observed may be due to other unidentified factors related to the institution where surgery was performed. It is true that in the previous study, we were not able to demonstrate specific nutritional deficiencies in the group of patients who developed PN after BS. However, the occurrence of PN after BS in the previous study was clearly associated with markers of poor nutritional status. Therefore the association of PN with malnourishment was undeniable.

Because the patients in our present cohort had more intensive, multidisciplinary nutritional evaluation and management than did patients in our previous cohort, the risk factors for developing postoperative PN were different. We no longer found that the amount of weight loss, serum albumin levels, and type of BS were risk factors. Patients who developed PN after BS attended nutritional clinics less frequently ($p<0.04$), but this variable ceased to be significant with use of univariate life-table analysis.

Two new risk factors identified in this cohort were increased glycosylated hemoglobin and triglyceride levels prior to BS. DM is known to predispose patients to various kinds of neuropathies, including mononeuropathy.¹⁰ Our previous study did not show DM to be a risk factor in PN after BS, however, in our current study, increased glycosylated hemoglobin proved to be a risk factor for PN. In a subgroup analysis in patients with mononeuropathies, increased glycosylated hemoglobin was also a significant risk factor. We believe that identification of increased baseline glycosylated hemoglobin as a risk factor confirms the observation of many other studies that the diabetic state predisposes to the development of neuropathy (including carpal tunnel syndrome). Hypertriglyceridemia has been reported to be associated with idiopathic painful neuropathy,^{11,12} although the mechanism has not been explained.¹³ Recently, the role of hyperlipidemia as a cause of peripheral neuropathy has been even more emphasized. Interaction between hyperlipidemia and hyperglycemia has been shown to cause oxidative stress to dorsal root ganglion neurons in diabetic patients.¹⁴ We found increased triglyceride levels in diabetic patients at follow-up to be a risk factor for PN after BS. Our findings suggest that an increased serum triglyceride level might not be an independent risk factor but rather a covariate with DM in the development of PN. Increased triglycerides are more common in type 2 DM.¹⁵ In our cohort, hypertriglyceridemia was associated with PN in diabetic patients only.

The reason that prolonged duration of hospital stay was a risk factor for PN, especially for mononeuropathy, is unclear. It is probable that prolonged hospital stay is usually found in patients who have more postoperative problems, need prolonged bed rest, and require more in-hospital procedures. All these factors can predispose a patient to mononeuropathy, often by compression or trauma, especially in patients who are experiencing rapid weight loss.

Prolonged GI symptoms, especially nausea and vomiting, and nutritional markers suggestive of ongoing malnutrition (such as decreased total lymphocyte count) were also risk factors for PN, and this emphasizes that ongoing nutritional surveillance is necessary in the postoperative period. Interestingly, the presence of postoperative complications that required hospitalization was a strong risk factor for developing PN only in diabetic patients. This

observation may mean that postoperative complications predispose a diabetic patient to developing PN or, alternatively, that patients with DM tend to get more complications after BS (a covariate). Protracted vomiting has been reported in association with vitamin B1 deficiency following bariatric surgery.¹⁶

In a cohort of patients who underwent BS at one institution and had a good maintenance of nutritional status with a high rate of attendance at a multidisciplinary nutritional clinic, the frequency of PN after BS is decreased, and mononeuropathies were the most frequent subtype. The risk factors for PN after BS in this setting are increased glycosylated hemoglobin at baseline, increased triglycerides at follow-up, the presence of prolonged postoperative GI symptoms, nausea and vomiting, and prolonged hospital stay. Most importantly, this study confirms that by optimizing nutritional status through attendance at a multidisciplinary nutritional clinic before and after BS, and by using good operative technique, the sensory-predominant type of polyneuropathy can largely be prevented.

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Abbreviations

BS	bariatric surgery
BMI	body mass index
DM	diabetes mellitus
GI	gastrointestinal
PN	peripheral neuropathy
TIBC	total iron binding capacity
TSH	thyroid stimulating hormone

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Table 1

Baseline Characteristics of Bariatric Surgery Patients

Continuous	Neuropathy				No Neuropathy				p *
	n	Mean	SD	Range	N	Mean	SD	Range	
Age, yr	26	46	11	26-74	367	43	11	16-71	0.22
Height, cm	26	169	10	148-192	367	169	10	142-196	0.62
Weight, kg	26	143	39	88-217	367	147	36	73-355	0.60
BMI, kg/m ²	26	50	13	31-87	367	51	11	30-101	0.65
Albumin, g/dl	21	4.1	0.3	3.3-4.7	273	4.0	0.3	2.8-5.2	0.14
Vitamin B12	2	274	187	142-406	59	475	304	155-2000	0.36
Folate	2	7.4	1.1	6.6-8.2	59	9.9	5.7	3.7-20.0	0.54
Hemoglobin	26	13	1	11-15	358	13	1	8-17	0.28
TLC×10 ³	25	2.0	0.8	0.4-3.8	344	1.9	0.8	0.3-5.1	1.00
Iron	3	64	27	34-88	54	60	33	1.2-159	0.86
TIBC	3	354	30	320-378	53	327	61	166-447	0.47
TSH	14	2.9	1.8	0.9-8.1	231	2.7	1.7	0.1-13.8	0.70
Cholesterol	21	216	48	142-342	320	207	56	108-715	0.47
Triglyceride	21	220	123	72-553	316	193	147	39-1555	0.40
Glucose	24	131	55	81-289	345	125	48	50-387	0.60
Glycosylated hemoglobin	9	11	3	6-15	127	7	2	4-14	<0.0001
Duration of hospital stay, days	16	11	12	7-56	305	9	3	4-25	0.01
Follow-up, mo	26	43	34	4-120	367	42	43	1-223	0.91
Dichotomous	n	Yes	%		N	Yes	%		p *
Sex, Female	26	19	73		367	265	72		1.00
DM	26	8	31		367	97	26		0.65
Type 1 DM	8	1	13		97	9	9		0.56
PO GI symptoms	24	6	25		365	24	7		0.01
PO Complications	24	2	8		365	9	2		0.14

Continuous	Neuropathy			No Neuropathy			p [*]
	n	Mean	SD	Range	N	Mean	SD
PO Nutritional clinic	24	19	79		359	332	92
PO Nutritional supplementation	24	20	83		358	321	90
PO Diarrhea	26	2	8		367	11	3
PO Dumping	26	0	0		367	2	1
PO Nausea and vomiting	26	3	12		367	11	3

SD = standard deviation; BMI = body mass index; TLC = total lymphocyte count;
TTBC = total iron binding capacity; TSH = thyroid stimulating hormone; DM = diabetes mellitus; PO = postoperative; GI = gastrointestinal.
^{*} T-test for continuous data and Fisher exact test for dichotomous data.

Table 2

Univariate Life-table Analysis of Risk Factors for PN after Bariatric Surgery

	Risk Factor	Hazard Ratio (95% confidence interval)	<i>p</i>
Risk factors for PN in all patients	Glycosylated hemoglobin at baseline	1.39 (1.14-1.71)	<0.01
	Triglyceride level at follow-up (per 10 mg/dL)	1.02 (1.00-1.04)	0.05
	Duration of hospital stay (per 10 days)	2.24 (1.19-4.24)	0.01
	Prolonged PO GI symptoms	3.04 (1.20-7.72)	0.02
	Prolonged PO nausea and vomiting	3.57 (1.07-11.9)	0.04
	Weight loss at follow-up	1.00 (0.94-1.01)	0.91
	Change in BMI at follow-up	1.01 (0.96-1.07)	0.69
	Albumin at follow-up	1.61 (0.37-6.93)	0.52
	Roux -en-Y gastric bypass	1.48 (0.57-3.87)	0.42
	Vertical banded gastroplasty	0.55 (0.18-1.65)	0.28
	Pancreatobiliary diversion	2.41 (0.56-10.33)	0.24
	PO nutritional clinic	0.58 (0.21-1.61)	0.29
Risk factors for Mononeuropathy subtype in all patients	Glycosylated hemoglobin at baseline	1.37 (1.02-1.83)	0.04
	Duration of hospital stay (per 10 days)	2.62 (1.39-4.94)	<0.01
Risk factors for PN in diabetic patients	Total lymphocyte count at follow-up	0.13 (0.02-0.69)	0.02
	Triglyceride level (natural log) at follow-up	4.60 (1.23-17.22)	0.02
	Glycosylated hemoglobin at baseline	1.37 (1.06-1.76)	0.01
	PO complication requiring hospitalization	15.02 (1.56-144.67)	0.02
Risk factors for PN in non-diabetic patients	Total lymphocyte count at follow-up	1.75 (1.04-2.95)	0.04
	Duration of hospital stay (per 10 days)	2.54 (1.37-4.71)	<0.01

PO = post operative.

Table 3

Patterns of Neuropathy in the Current and Previous Cohorts

	Current Cohort n=393	Previous Cohort n=360*	<i>p</i> **
Peripheral Neuropathy (%)	26 (7%)	45 (13%)	<0.01
Mononeuropathy (%)	21 (5%)	18 (5%)	0.83
Sensory-predominant polyneuropathy (%)	2 (1%)	25 (7%)	<0.0001
Radiculoplexus neuropathy (%)	3 (1%)	2 (1%)	0.73

* Previous cohort n=435 but we excluded 75 patients who were also in the current cohort.

** Chi squared test.